Here's how to recover from nicotine:

* **Understand the withdrawal symptoms:** Nicotine withdrawal can cause a range of unpleasant symptoms, including cravings, irritability, anxiety, difficulty concentrating, and sleep disturbances. Understanding these symptoms can help you prepare for them and manage them effectively.

* **Set a quit date:** Choose a specific date to stop using nicotine products. This gives you a clear goal to work towards and helps you mentally prepare for the transition.

* **Develop a support system:** Talk to your friends, family, or a healthcare professional about your

decision to quit. Having a strong support network can provide encouragement and help you stay on track.

*** **Consider nicotine replacement therapy (NRT):** NRT products, such as patches, gum, lozenges, inhalers, and nasal sprays, can help reduce cravings and withdrawal symptoms by providing controlled doses of nicotine without the harmful chemicals found in tobacco or e-cigarettes. Consult with a healthcare professional to determine if NRT is right for you and which product would be most effective.**

*** **Explore prescription medications:** Certain medications, such as bupropion (Zyban) and varenicline (Champix), can help reduce cravings and withdrawal symptoms by affecting brain chemicals involved in nicotine**

addiction. These medications require a prescription from a healthcare professional.

* **Practice relaxation techniques:** Stress and anxiety can worsen withdrawal symptoms. Incorporate relaxation techniques into your daily routine, such as deep breathing exercises, meditation, yoga, or spending time in nature.

* **Stay active:** Regular physical activity can help reduce cravings, improve mood, and boost overall well-being during the recovery process. Aim for at least 30 minutes of moderate-intensity exercise most days of the week.

* **Avoid triggers:** Identify situations, places, or people that trigger your nicotine cravings and try to avoid them as much as possible. If you can't avoid them entirely,

develop strategies for coping with the cravings when they arise.

* **Seek professional help:** If you're struggling to quit on your own, consider seeking professional help from a healthcare provider, therapist, or smoking cessation program. They can provide personalized guidance, support, and resources to help you succeed.

* **Be patient and persistent:** Quitting nicotine is a challenging process that may require multiple attempts. Don't get discouraged if you experience setbacks along the way. Learn from your experiences and keep trying until you achieve your goal.

Using the Recovery Kernel Framework

Cigarettes · Vaping / E-Cigarettes · Nicotine Pouches · Snus · Chewing Tobacco · Hookah · Cigars · Pipes

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 000 or text 13 11 14

Nicotine is the most globally prevalent addiction. The medicine exists. The biology is understood. Quitting is possible for every person who wants to — with the right support.

Een bericht voordat u begint.

1.3 billion people smoke tobacco worldwide. More people are addicted to nicotine than to every other substance in this guide series combined. Tobacco kills approximately 8 million people annually — more than alcohol, opioids, stimulants, and every other recreational drug added together. And nicotine addiction is one of the most successfully treated substance use disorders in medicine, with multiple evidence-based pharmacological options, clear genomic predictors of response, and cessation rates that improve dramatically with the right combination of tools.

The gap between the scale of the problem and the sophistication of the treatment conversation is extraordinary. People who would never consider treating opioid withdrawal without buprenorphine will attempt nicotine withdrawal with willpower alone — and fail, repeatedly, concluding that they cannot quit rather than that they are using inadequate tools.

Il compendio, nella sezione dedicata ai farmaci GLP-1, cita direttamente tabacco e nicotina: "numerose segnalazioni aneddotiche di cessazione spontanea del fumo o riduzione; studi clinici in corso a partire dal 2026". Lo stesso meccanismo che riduce il segnale di desiderio di ricompensa per alcol e oppioidi sta causando la cessazione spontanea nei fumatori dipendenti da nicotina che assumono semaglutide. La dipendenza dalla nicotina è un problema di neurobiologia con soluzioni farmaceutiche; lo stesso quadro applicabile a tutte le altre sostanze in questa serie si applica anche qui, con una base di evidenze ancora più ampia.

This guide covers every delivery form—cigarettes, vaping, nicotine pouches, snus, chewing tobacco, hookah—because the nicotine is the addiction regardless of the delivery mechanism, and the delivery mechanism determines the specific physical health risks being accumulated alongside the nicotine dependence.

De genetische informatie is duidelijk. De medicijnen zijn beschikbaar en effectief. De biologie van herstel is begrepen. Deze handleiding biedt een compleet kader voor het gebruik van al deze kennis.

Section 1 — What Nicotine Did to Your Brain

THE CENTURION — The most efficient dopamine delivery system available over any pharmacy counter.

Nicotine reaches the brain within 10 seconds of inhaling a cigarette. This speed of delivery is faster than intravenous cocaine and comparable only to smoked crack in the speed of its CNS impact. The addiction mechanism is clear and well-documented: nicotine activates nicotinic acetylcholine receptors (nAChRs) in the ventral tegmental area (VTA), producing dopamine release in the nucleus accumbens.

The most important nicotinic receptor subtype for addiction: the α4β2 nAChR. Nicotine's activation of this receptor is the primary driver of the dopamine release that produces cigarettes' rewarding properties. Chronic nicotine exposure downregulates α4β2 receptors — and then withdrawal deprives those receptors of the nicotine signal they have come to require, producing the withdrawal state.

1.1 — The Multi-System Impact of Tobacco

Nicotine's acute effects include: * **Increased heart rate and blood pressure** * **Stimulation of alertness** * **Appetite suppression** * **Release of dopamine, creating a sense of pleasure** * **Potential for addiction**	**Tobacco smoke adds:** * **Tar** * **Nicotine** * **Carbon monoxide** * **Other chemicals**	**What chronic use produces?**
nAChR-aktivierung → Dopaminfreisetzung → Belohnung und Verstärkung.	4,000+ chemical compounds including MAO inhibitors (harman, norharman) that block dopamine breakdown — making tobacco smoke MORE rewarding than nicotine alone. This is why NRT doesn't fully replicate smoking.	nAChR upregulation (paradoxically – more receptors to compensate for chronic activation); dopamine system adapted to constant nicotine input; baseline mood and concentration require nicotine to reach 'normal'.
Cognitive sharpening —	Monossido de carbono (CO) deslocando o oxigênio na	La capacità cognitiva diventa veramente dipendente dalla

improved attention, working memory, processing speed.	hemoglobina. A paradoxa: o fumante sente-se mais alerta, apesar da redução do fornecimento de oxigênio para o cérebro.	nicotina: la sospensione provoca deficit misurabili nell'attenzione e nella memoria di lavoro che si manifestano come un deterioramento, ma sono in realtà la rimozione di un potenziamento farmacologico.
Anxiolytic effect — reduces acute anxiety through nAChR-mediated GABA modulation.	Tobacco's MAO inhibitors also affect serotonin metabolism — contributing to the mood stabilisation smokers report from cigarettes in ways pure nicotine does not match.	The smoker develops a genuine pharmacological anxiety management mechanism. Withdrawal produces anxiety because the mechanism is removed. The anxiety in early cessation is real withdrawal, not character weakness.
Appetite suppression — nicotine activates POMC neurons in the hypothalamus.	Tobacco's effect on metabolism and gut motility contributes to appetite suppression, in addition to nicotine's direct mechanism.	Gain de poids moyen de 4 à 8 livres est un effet secondaire pharmacologique attendu et une réponse métabolique normale lors de l'arrêt. Gérer l'anxiété liée à cette prise de poids fait partie du processus d'arrêt.

1.2 — The Tobacco Product Landscape

Product	Velocità di rilascio della nicotina.	Unique physical risks.	Cessation considerations.
Cigarettes.	Fastest — 10 seconds to peak brain nicotine. 8–20 cigarettes per smoking session in heavy smokers.	Cancer du poumon, BPCO, maladies cardiovasculaires, accident vasculaire cérébral. 50 % des fumeurs décèdent de maladies liées au tabac.	Full MAT range applies. Most studied cessation population. Varenicline is the most effective single medication.
E-cigarettes / vaping / JUUL	Fast — comparable to cigarettes for modern pod devices. Very high nicotine concentration in salt form.	EVALI (lesão pulmonar associada ao uso de cigarros eletrônicos ou produtos para vaporização) causada pelo acetato de vitamina E em alguns produtos. Os riscos a longo prazo são menos conhecidos	Vaping might assist with quitting cigarettes, but it can create its own nicotine dependence. The aim of using vaping as a smoking cessation aid is to ultimately stop vaping altogether. Full Medication-Assisted Treatment (MAT) applies.

		do que os dos cigarros. Formadeído em altas temperaturas.	
Nicotine pouches (Zyn, On!, Velo)	Slower — absorbed through the oral mucosa over 30–60 minutes. No combustion products.	Gum disease and oral tissue irritation with long-term use. No combustion products — significantly lower cancer risk than cigarettes.	Lower immediate health risk than cigarettes, but still produces nicotine dependence. Can be useful as a cessation step-down if used as part of a planned reduction.
Snus (tabaco sueco para mascar).	Moderate — oral mucosal absorption. Slower than cigarettes.	Increased risk of pancreatic cancer. Lower risk of oral cancer compared to chewing tobacco. Cardiovascular risk. No lung disease from non-combustion.	Snus wordt gebruikt als een methode om de schadelijke gevolgen van sigaretten te verminderen in Zweden (veel lagere sterfte door tabak dan in andere Europese landen). Dezelfde afhankelijkheid van nicotine vereist dezelfde aanpak voor het stoppen.
Chewing tobacco / dip / snuff.	Lenta absorción a través de la mucosa oral durante un período prolongado.	Cancer of the mouth, throat, and esophagus; gum disease; tooth loss; leukoplakia (changes in oral tissue). No lung disease.	Rates of quitting are lower compared to those seen with cigarettes, according to published research. Nicotine replacement therapy (NRT) patches and gum are especially important because the act of putting something in your mouth is a habit in itself, and this habit needs its own type of help to change it.
Hookah/shisha	Lenta doziranje tijekom svake sesije, ali trajanje sesije je dugo (45-60 minuta). Ukupna izloženost nikotinu i ugljičnom monoksidu tijekom jedne sesije može premašiti količinu u paketi cigareta.	Risque d'intoxication au monoxyde de carbone provenant du tabac chauffé au charbon. La fumée secondaire est importante. Tous les risques de cancer liés au tabagisme.	L'uso di narghilè viene spesso sottovalutato perché considerato "meno dannoso" a causa della sua natura sociale e intermittente. Tuttavia, l'esposizione totale ai composti tossici è elevata durante ogni sessione. L'approccio alla cessazione deve essere lo stesso utilizzato per le sigarette.

1.3 — The Psychiatric Comorbidity Pattern

THE CENTURION — Smoking Is Not Random — It Concentrates in the Most Neurobiologically Vulnerable Populations.

The compendium's framework of substance use as a signal pointing to underlying biology is nowhere more clearly demonstrated than in tobacco. Smoking rates in the general population: approximately 12–15% in developed countries. Smoking rates in psychiatric populations:

- Schizophrenia and schizoaffective disorder: 70–90%. People with schizophrenia are using nicotine's nAChR agonism to modulate the hypodopaminergic state that the illness and its antipsychotic treatment produce. They are self-medicating with a degree of biological precision that the recovery framework must acknowledge.
- Bipolar disorder: 40–70%. Nicotine's mood-stabilizing effects (through nAChR-mediated serotonin and dopamine modulation) serve a genuine mood regulation function that cessation removes.
- ADHD: 35–40%. Nicotine's cognitive-sharpening effects through nAChR activation in the prefrontal cortex directly address ADHD's dopaminergic deficit. Nicotine is ADHD self-medication.
- Major depressive disorder: 40–60%. The MAO inhibitors in tobacco smoke provide genuine antidepressant effects that pure nicotine does not replicate — making tobacco cessation harder than nicotine cessation for people with depression.
- Alcohol use disorder: 70–80%. The stimulant-depressant interaction, shared dopamine circuitry, and social co-conditioning produce the highest smoking-alcohol co-occurrence in addiction medicine.

For many people, quitting smoking requires addressing any underlying mental health conditions that smoking may have been masking. Quitting without psychological support often doesn't work in these cases – not due to a lack of determination, but because a vital coping mechanism has been taken away without providing an alternative.

Section 2 — Your Genomic Blueprint

Nicotine addiction has one of the most thoroughly characterized pharmacogenomic profiles in all of addiction medicine. The CHRNA5/CHRNA3/CHRNB4 gene cluster is the strongest genetic predictor of nicotine dependence identified in genome-wide association studies. CYP2A6 determines how fast nicotine is cleared — with direct implications for MAT selection and dosing.

The DNA Testing Panel for Nicotine Recovery.

Gene	What it controls.	Key variant.	Nicotine recovery relevance.
CHRNA5 / CHRNA3 / CHRNB4	Nicotinic acetylcholine receptor subunits α5, α3, and β4 — the primary receptor complex mediating nicotine's effects in the central nervous system (CNS) and peripheral nervous system.	rs16969968 (CHRNA5) — A allele (risk); rs1051730 (CHRNA3) — T allele; multiple CHRNB4 variants.	THE primary nicotine vulnerability gene cluster. Individuals carrying the A allele at rs16969968: smoke more cigarettes per day, have higher nicotine dependence severity scores, find it harder to quit, and have a higher risk of lung cancer (independent of the amount smoked — the receptor variant itself appears to increase lung cancer risk). They also respond BETTER to varenicline (the partial nAChR agonist) than to NRT alone. This is clinically actionable: if you carry the A allele at rs16969968, varenicline is your most effective cessation tool.
CYP2A6	Metabolismo primario de la nicotina: convierte la nicotina en cotinina para su eliminación. También metaboliza las nitrosaminas (carcinógenos del tabaco).	*2, *4, *12: varianti di metabolismo lento; *1B: variante di metabolismo rapido; varie varianti intermedie.	THE most clinically important gene for NRT dosing. Slow CYP2A6 metabolizers: nicotine is cleared more slowly — they require fewer cigarettes per day to maintain nicotine levels, they get more nicotine per cigarette, and they appear to have an EASIER time quitting because the slower clearance means withdrawal is more gradual. They also have LOWER lung cancer risk — the slower metabolism of carcinogens is protective. Slow metabolizers: NRT patches may be more effective than gum (slower, more sustained delivery matching their slower clearance). Fast metabolizers (CYP2A6*1B): faster

			clearance means more cigarettes needed, harder time quitting, higher cancer risk, better Response to higher-dose NRT.
COMT	Clearance de la dopamine : détermine l'importance de la récompense dopaminergique du tabac.	Val158Met (rs4680) — Val/Val Warrior.	Warrior COMT (Val/Val, fast dopamine clearance) is overrepresented in heavy smokers — nicotine's dopamine release is particularly valued when dopamine clears rapidly at baseline. Warrior constitution recovers from nicotine's dopamine contribution faster per cigarette but also needs that contribution more. Recovery architecture: the Warrior's natural intensity and dopamine needs must be met through high-stimulation natural rewards alongside MAT.
MTHFR	Metilazione — sintesi di serotonina e dopamina.	C677T, A1298C	MTHFR variants contribute to the depression and anxiety that tobacco was managing. The antidepressant-like effects of tobacco's MAO inhibitors are particularly valued by people with MTHFR-impaired monoamine synthesis. 5-MTHF supplementation can address the underlying synthesis deficit and often reduces the mood component of cessation difficulty significantly.
DRD2 / ANKK1	D2 receptor density — Reward Deficiency Syndrome.	Taq1A (rs1800497) A1 allele.	A1 allele (reduced D2 density, RDS) is associated with heavier smoking and greater difficulty quitting — the baseline reward deficit means nicotine's dopamine release is more essential to normal functioning. Recovery: the Brain Stack (UMP + DHA + CDP-Choline) for D2 receptor upregulation is a meaningful parallel intervention for the RDS profile.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression — anxiety sensitivity.	Short/short allele.	Short/short carriers with high baseline anxiety find cigarettes' nAChR-mediated anxiolytic effects particularly compelling. Cessation produces genuine anxiety increase for these individuals. The most effective cessation approach for short/short SLC6A4: varencline + buspirone for anxiety management + SSRI or 5-MTHF for the underlying serotonin deficit. Addressing the anxiety is the central challenge.

Fenotipificación sin pruebas.

- If you smoked more than a pack a day and found it significantly harder to quit than your peers, you likely have the CHRNA5 rs16969968 A allele. Varenicline is your best medication (MAT) option.
- Do you smoke fewer cigarettes but find each one feels very satisfying and necessary? → Possible CYP2A6 slow metabolizer. An NRT patch (provides a steady dose) may suit you better than chewing gum.
- Did you start smoking for the focus benefit, and do you also struggle with attention? → DAT1/DRD4 ADHD profile. ADHD evaluation alongside cessation planning.
- Did smoking reliably reduce your anxiety, and do you find that stopping smoking causes severe anxiety? → SLC6A4 short/short. Anxiety management is a primary focus of your cessation plan.
- Did smoking help with depression and do previous quit attempts produce severe depressive episodes? → MTHFR + tobacco MAO inhibitor dependence. 5-MTHF first, bupropion as cessation aid, psychiatric monitoring during cessation.

Section 3 — Medication-Assisted Treatment: The Most Effective Cessation Tools.

THE CENTURION — La combinazione di Terapia Sostitutiva della Nicotina (NRT) e Vareniclina è ora l'intervento singolo più efficace per smettere di fumare, più efficace di qualsiasi approccio basato sulla forza di volontà.

The evidence base for nicotine cessation pharmacotherapy is more robust than for almost any other substance use disorder. Multiple large clinical trials across decades have produced clear, actionable guidance. The person who attempts nicotine cessation without medication support is statistically likely to fail — not because they lack commitment but because they are attempting an unnecessarily difficult neurobiological task without available pharmaceutical assistance.

3.1 — The Medication Options

Medication.	Mechanism.	Improvement in cessation rates.	Key considerations
Varenicline (Chantix / Champix)	Partial α4β2 nAChR agonist — activates the receptor (reducing withdrawal) while blocking nicotine's full agonist effect (reducing reward from smoking). The most pharmacologically specific cessation medication.	2–3 times more effective than a placebo. The most effective single smoking cessation medication based on current research. Individuals with the CHRNA5 A allele tend to respond best to this medication.	The psychiatric safety warning was revised in 2016 — current evidence shows no significantly elevated psychiatric risk at standard doses. DOES cause vivid dreams (nAChR modulation affects sleep architecture). CYP450 not significantly involved — dosing is less affected by CYP variants than other medications.
Combination NRT (patch + gum or lozenge).	Nicotine replacement at lower doses than smoking, tapering over weeks. The patch provides a steady baseline; the gum/lozenge provides acute "rescue" for craving spikes.	1.5–2.5 gånger mer effektivt än placebo. Kombination av nikotinersättningsbehandling (NRT) är betydligt mer effektiv än enstaka NRT.	CYP2A6 slow metabolizers: the patch may be more effective (slower, steadier delivery matching their metabolism). CYP2A6 fast metabolizers: may need higher patch doses and more frequent gum use. Gum: chew slowly, place between cheek and gum, do not swallow — the nicotine is absorbed

			sublingually.
Bupropion (Zyban / Wellbutrin)	Inhibition of dopamine and norepinephrine reuptake; also an nAChR antagonist at certain receptor subtypes. Functions as both a smoking cessation aid and an antidepressant, addressing co-occurring depression and smoking simultaneously.	1.5–2x more effective than placebo. Particularly effective for smokers with comorbid depression.	CYP2B6 metabolized — CYP2B6 variants affect plasma levels. Also, the medication is affected by CYP2D6 inhibition. Note: bupropion lowers the seizure threshold — contraindicated in people with eating disorders, seizure history, or current alcohol withdrawal. Most effective for MTHFR-associated depression-driven smoking when 5-MTHF is not adequate.
Combination Varenicline + NRT (highest evidence approach).	The combination of varenicline (a partial agonist that reduces reward and withdrawal) with an NRT patch (providing a steady level of nicotine replacement) is more effective than either treatment used alone.	3–4 lần hiệu quả hơn so với giả dược trong một số thử nghiệm—tỷ lệ cai thuốc cao nhất trong các nghiên cứu hiện có.	This combination is underutilised because healthcare providers often present medications as alternatives rather than additive options. Both can be used simultaneously. Discuss with prescriber.
GLP-1 agonists (semaglutide / tirzepatide — emerging).	The compendium: "multiple anecdotal reports of spontaneous smoking cessation or reduction; clinical trials ongoing as of 2026." GLP-1 receptors in the nucleus accumbens and VTA dampen the dopaminergic wanting signal — reducing the compulsive drive to smoke.	Klinische Studien laufen noch. Anekdotische Berichte deuten darauf hin, dass die Absetzraten bei einigen Anwendern von GLP-1-Agonisten zur Behandlung metabolischer Erkrankungen vergleichbar mit oder höher als bei Varenicline sein könnten.	Not yet FDA-approved specifically for nicotine cessation, but the emerging evidence is significant. Discuss with your doctor if you are taking GLP-1 agonists for other conditions, as smoking cessation may occur as a beneficial side effect.
Cytisine (Tabex – available in Eastern Europe; *Cytisus laburnum* derivative).	Natural plant-derived partial nAChR agonist — same mechanism as varenicline but discovered first and used in Eastern	Comparable effectiveness to varenicline in head-to-head trials. Available as a supplement or over-the-counter	Less readily available in the US and UK, but accessible online. The most cost-effective smoking cessation option when the cost of varenicline is prohibitive.

	Europe for decades. Now being studied for Western markets.	medication in some countries.	

3.2 — The Cessation Strategy Framework

THE CENTURION — La medicación es la base, no toda la estructura.

The most evidence-based cessation approach combines: (1) pharmacological support from the medications above, (2) behavioral support through cessation counseling or a structured program, and (3) the biological recovery support in Section 4. Each component addresses a different dimension of the addiction:

- Pharmacological: reduces the severity of withdrawal symptoms and the rewarding effects of smoking, making it easier to quit from a neurological perspective.
- Behavioral: identifies triggers, builds coping strategies, addresses the ritual and habitual dimensions of smoking behavior (the coffee-cigarette pairing, the post-meal cigarette, the stress-cigarette reflex).
- Biological recovery: rebuilds the dopamine, serotonin, and nAChR systems that nicotine has adapted, providing the neurochemical substrate for genuine comfort in cessation.

The quit date approach: Set a specific quit date 1–2 weeks in advance. Begin varenicline or bupropion 1 week BEFORE the quit date — the medication needs time to reach effective plasma levels. NRT begins ON the quit date. Having the medication already at therapeutic levels on the quit date dramatically improves outcomes.

The gradual reduction approach: For people who cannot commit to a quit date: structured reduction over 12 weeks with NRT as a bridge — reducing cigarette count by 50% in the first 4 weeks, 75% by week 8, quit by week 12. Less studied than the quit date approach but may improve engagement.

The dual use period: Using NRT or varenicline while still smoking is not failure — it is a harm reduction phase for people in the approach stage. Combination NRT during a 'pre-quit' period is associated with higher ultimate cessation rates.

Section 4 — Biological Reconstruction

Quitting nicotine causes withdrawal symptoms that are genuinely biological — not character weakness. The nAChR upregulation caused by chronic nicotine use means the receptors are now undersupported by their natural ligand (acetylcholine). The MAO inhibitors found in tobacco smoke are no longer present. The dopamine release triggered by each cigarette is absent. The biological reconstruction protocol supports the recovery of all three systems.

4.1 — The Methylation Foundation

- 5-MTHF (400–1000mcg, morning): supports serotonin and dopamine synthesis. For MTHFR-variant smokers who were managing depression through tobacco's MAO inhibitors, 5-MTHF provides the synthesis support that can partially replace the mood-stabilizing function.
- Methylcobalamin (1000–2000mcg sublingually, in the morning): supports methylation, particularly relevant for smokers who have been consuming nicotine-associated oxidants that deplete B12.
- P5P (25–50mg, morning): cofactor for monoamine synthesis pathways that tobacco's MAO inhibitors were enhancing.
- Magnesium glycinate (400–600mg elemental, taken in the evening): helps reduce anxiety; supports GABA function; improves sleep – addresses all three major areas of withdrawal symptoms.

4.2 — nAChR System Restoration

THE CENTURION — Supporting the receptor system that was calibrated around nicotine.

The nicotinic acetylcholine receptor system needs time to downregulate back toward its pre-smoking density and sensitivity. This process takes 4–6 weeks for the nAChR population to substantially recover. The following supports this recovery:

- Alpha-GPC (300–600mg, morning): choline precursor for acetylcholine synthesis. As the nicotinic receptor system readjusts to relying on endogenous acetylcholine rather than exogenous nicotine, supporting acetylcholine synthesis helps maintain the cognitive and mood functions that nicotine was enhancing.
- CDP-Choline (Citicoline, 250–500mg, morning): a combined choline and cytidine source. Supports both acetylcholine synthesis and dopamine receptor recovery—addressing the nAChR and dopamine aspects of nicotine withdrawal simultaneously.
- Lion's Mane mushroom (500–1000mg, daily): Stimulates NGF and BDNF. Nicotine has documented neuroprotective effects through nAChR activation

of neurotrophic factors. Lion's Mane provides NGF stimulation through a different pathway, partially replacing this neuroprotective function.

- Huperzine-A (50–100mcg, daily — cycle with breaks): acetylcholinesterase inhibitor, slowing acetylcholine breakdown. Supports cognitive function during the period when acetylcholine activity is reduced by the absence of nicotinic potentiation. Use on alternating weeks to prevent tolerance.

4.3 — Dopamine and Mood Recovery.

- L-Tyrosine (500–1000mg, morning, away from protein): dopamine precursor. The dopamine contribution of nicotine's nAChR activation is significant — tyrosine supports the dopamine system as it re-establishes independent function.
- NAC (600–1200mg, daily, divided): helps with glutamate balance and reduces cravings. There's evidence for this specifically in quitting smoking. Start taking it right away when you quit.
- Rhodiola rosea (300mg, morning): modulates dopamine and serotonin; adaptogen; anti-fatigue. The motivation and energy deficit experienced during the initial stages of quitting smoking can be addressed with Rhodiola's mild dopaminergic effects.
- 5-HTP (50–100mg, evening): helps rebuild serotonin levels to improve mood and reduce anxiety. MAO inhibitors in tobacco products can have antidepressant effects; 5-HTP supports serotonin recovery as that mechanism is removed. Do not combine with SSRIs.

4.4 — The Weight Gain Management

THE CENTURION — 4–8 lbs. Expected, metabolically normal, and not a permanent state.

Nicotine suppresses appetite through hypothalamic POMC neuron activation and increases metabolic rate slightly. Cessation removes both effects — weight gain of 4–8 lbs on average is the biological consequence.

- This weight gain is not related to eating more — it occurs even with controlled caloric intake. It is the metabolism normalizing from nicotine-enhanced levels.
- The weight gain is most pronounced in the first 3 months and stabilizes or reverses with exercise and time.
- Do not use weight gain anxiety as a reason to continue smoking — the cardiovascular and cancer risk of smoking far exceeds the health risk of 4–8 lbs.
- Exercice, en particulier l'exercice aérobie décrit dans la section 5, est le moyen de gestion du poids le plus efficace pendant l'arrêt du tabac ET fournit

un remplacement de la dopamine et des endorphines qui soutient simultanément l'arrêt.

- GLP-1 agonists (semaglutide, tirzepatide): For individuals where concerns about weight gain represent a major obstacle to quitting, GLP-1 agonists can help by addressing both the smoking cessation support and the weight management issue at the same time. Discuss with your doctor.

4. 5 — Sleep Reconstruction

Nicotine disrupts sleep through nAChR-mediated arousal, yet paradoxically many smokers report that cigarettes help them sleep. This reflects the withdrawal anxiety that a cigarette resolves before bed — the cigarette is managing nicotine withdrawal rather than producing sleep. Cessation initially produces vivid dreams (nAChR recovery producing REM rebound) and sleep fragmentation that resolves within 2–4 weeks.

- Melatonin (0.5–1mg sublingual, taken at the same time each day): helps regulate your body clock during periods of disrupted sleep.
- Magnesium glycinate (400mg at night): the main way to manage anxiety related to quitting, which can make it hard to fall asleep.
- Glycine (3–5g, 30 minutes before bed): reduces core body temperature to aid sleep onset.
- L-Theanine (200mg evening): promotes alpha waves; reduces the cognitive hyperactivity associated with nicotine withdrawal.

Varenicline's vivid dream side effect is caused by a change in sleep architecture mediated by nAChRs. This is not dangerous, resolves over time, and is manageable for most people. If severe: taking varenicline earlier in the day (not with an evening meal) can reduce the impact on sleep.

4.6 — Pulmonary Recovery

- Within 20 minutes: blood pressure and heart rate normalize.
- Within 12 hours: carbon monoxide cleared from blood; oxygen-carrying capacity normalized.
- Within 2 weeks: circulation improving; lung function improving measurably.
- Within 1 year: cardiovascular disease risk halved.
- Within 5 years: stroke risk comparable to non-smoker.
- Within 10 years: lung cancer risk halved.

N-Acetyl Cysteine (NAC 600mg twice daily): mucolytic — helps clear accumulated mucus and reduces oxidative stress from decades of smoke exposure. Particularly valuable in the first months of cessation. The compendium names NAC and glutathione as specifically depleted by cigarettes/alcohol — replenishment is a genuine recovery support.

- Vitamina C (1000–2000 mg diariamente): el humo del cigarrillo agota drásticamente la vitamina C. Los fumadores necesitan una ingesta de vitamina C significativamente mayor que los no fumadores, incluso después de dejar de fumar, ya que la capacidad antioxidante se recupera.
- Vitamin E (400 IU, mixed tocopherols): fat-soluble antioxidant for pulmonary membrane repair.
- EGCG (ekstraksi teh hijau, 400mg): antioksidan dengan sifat pelindung khusus pada jaringan paru-paru; mengurangi stres oksidatif akibat residu asap tembakau.

Section 5 — The Nicotine Craving — Understanding and Interrupting.

THE CENTURION — The Three-Minute Craving — and the Architecture That Makes It Manageable.

Nicotine cravings have a specific and documented temporal profile: the peak intensity of a nicotine craving lasts 3–5 minutes. Not 30 minutes. Not an hour. Three to five minutes. Every nicotine craving in the first 2–4 weeks of cessation will peak and subside within five minutes if it is not acted upon.

This is the most clinically useful single piece of information in nicotine cessation. The craving feels like it will go on forever. It will not. It will peak in under three minutes and then decline. The entire intervention has to do is outlast three minutes.

The Nicotine Craving Trigger Map.

Tipo di trigger	The conditioned pattern.	The interrupt.
Habitual/temporal cues (coffee, after meals, morning wake-up).	The most powerful nicotine triggers are temporal and habitual — the morning coffee is inseparable from the cigarette because they were paired thousands of times. The craving fires at coffee, not from physiological need at that moment.	Comportamiento disruptivo: cambiar la secuencia habitual. Tomar café en un lugar diferente. Caminar después de comer en lugar de quedarse parado donde se fumaba. El ciclo del hábito interrumpido: identificar el estímulo, cambiar la rutina que le sigue y mantener la recompensa mediante una sustitución.
Stress and anxiety triggers: * Workplace stress * Financial difficulties * Relationship problems * Health concerns * Major life changes * Traumatic events * Social isolation * Lack of sleep * Poor diet * Substance abuse	Nicotine's anxiolytic effect means that every significant stressor was paired with a cigarette as the coping response. The stress → cigarette pathway is deeply conditioned and the most common cause of relapse.	4-7-8 breathing is the most direct replacement for this — activating the same parasympathetic pathway that nicotine's nAChR activation provides. Magnesium. L-Theanine. The craving in this context is pointing at real stress that needs managing — address the stress, not just the craving.
Social triggers —	Social conditioning from years of	Inform your social contacts

being around smokers.	shared smoking creates powerful cue reactivity in social contexts — particularly bars, parties, work breaks, and any social situation where smoking was a shared activity.	about your decision to quit. Request that people refrain from smoking in your presence, especially during the early stages of quitting. Keep nicotine replacement therapy (NRT) readily available, such as gum or lozenges, for use in social situations. The social environment can be particularly challenging because it combines cue-reactivity with social pressure.
The false permission moment.	La negociación interna: "He tenido un día difícil. Me merezco un cigarrillo. Solo uno no lo arruinará todo". Este patrón cognitivo es el precursor más común de una recaída completa, no un desencadenante externo, sino una racionalización interna.	The compendium's precision language principle: "I am rationalizing returning to a neurologically addictive behavior because my dopamine system is depleted and is generating a motivation to restore itself." Name the mechanism. One cigarette WILL restart the conditioning — this is not abstinence perfectionism but pharmacological reality.

THE NICOTINE CRAVING INTERRUPT — 3-MINUTE PROTOCOL.

1. Name the craving and set a timer. The craving will be gone or substantially diminished when the timer ends. This is not a metaphor—it is the documented temporal profile of a nicotine craving.
2. EXECUTE 4-7-8 BREATHING — FIVE CYCLES. The anxiolytic function of nicotine is partly the parasympathetic activation of the smoking breath itself — the long exhale of blowing smoke. 4-7-8 extended exhale provides the same vagal activation without the nicotine.
3. USE NRT RESCUE. Nicotine gum or lozenge provides rapid (10–15 minute) nicotine delivery that arrests the withdrawal craving. NRT is not failure — it is the medication working as intended. Use it.
4. HYDRATE. Drink 470ml of water immediately. Dehydration worsens withdrawal irritability and the physical discomfort caused by nicotine withdrawal.
5. CHANGE PHYSICAL LOCATION. Move away from the triggering environment. Walk outside. The craving's contextual component is disrupted by physical environment change.
6. Wait. The timer is expiring. The craving has peaked. It is declining.

Section 6 — Building the natural architecture for what nicotine provided.

THE CENTURION — The Four Functions Cigarettes Were Serving — Rebuilt Without the Carcinogens.

Every cigarette served multiple functions simultaneously — dopamine reward, anxiety management, cognitive sharpening, social belonging, stress ritual. The natural recovery architecture addresses each.

The Functional Replacement Map.

The function of nicotine includes: * **Acting as a stimulant.** * **Binding to nicotinic acetylcholine receptors.** * **Affecting dopamine release.** * **Having addictive properties.**	**The natural replacement.**	**Implementation.**
Dopamine and reward.	Exercise (most powerful available dopamine replacement), cold exposure, achievement, creative work, competition.	Daily aerobic exercise: 30–45 minutes. Cold shower finale. High-intensity intervals. Something that challenges your skill level. The dopamine must be replaced—without replacement, the baseline flatness of nicotine withdrawal is genuine and persistent.
Anxiety management and stress relief.	4-7-8 breathwork (daily practice becomes the stress response), magnesium, exercise, meditation, cold exposure.	La respiración 4-7-8 debe convertirse en algo tan automático como alcanzar un cigarrillo. Mínimo de 10 minutos por la mañana y 10 minutos por la noche. El acto físico de practicar la respiración reemplaza el acto físico de fumar como un

		comportamiento para interrumpir el estrés.
Cognitive sharpening — focus and mental clarity.	Alpha-GPC + huperzine (support for acetylcholine), exercise (increases BDNF and norepinephrine), omega-3 DHA (supports cell membranes), ADHD treatment if indicated.	The cognitive clarity resulting from nicotine's nAChR activation takes 4–6 weeks to be replaced naturally. The supplement regimen described in Section 4.2 helps support this transition. For individuals with ADHD: the ADHD treatment provides a long-term alternative.
Social belonging — shared ritual, work break structure, community.	Finding social ritual replacements: walking meetings instead of smoking breaks, non-smoking social contexts, a recovery community that provides genuine belonging.	The social function of taking a cigarette break is often underestimated. Schedule non-smoking social time deliberately. Walk with colleagues instead of smoking. The sense of belonging was genuine; it simply requires finding that same feeling in a different setting.
Stabilizzazione dell'umore: la funzione antidepressiva degli inibitori della monoamino ossidasi (MAO) del fumo di tabacco.	5-MTHF for MTHFR-variant depression; exercise (most evidence-based natural antidepressant); bupropion as a cessation aid that also addresses the mood dimension; direct psychiatric treatment if clinical depression is present.	For people whose mood significantly worsened with previous quit attempts: this is not coincidence or weakness. Tobacco's MAO inhibitors were providing genuine antidepressant effects. Address this dimension pharmacologically through bupropion (which inhibits dopamine/norepinephrine reuptake and has MAO inhibiting properties) or through direct psychiatric treatment.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map.

Fase.	Protocols	Primary focus.	Nicotine-specific notes
Pre-Quit (1–2 weeks before quit date)	Prescriber; CYP2A6 and CHRNA5 testing if available; Section 3.	Start taking varenicline or bupropion; have nicotine replacement therapy (NRT) ready for your quit date; identify your triggers; plan ways to disrupt your usual behaviours.	Medication must be started BEFORE the quit date. Varenicline requires a 1-week pre-quit initiation. Bupropion requires 1–2 weeks. NRT begins ON the quit date. The pre-quit period sets the medical foundation.
Quit Date Onward (Weeks 1–4)	Protocol 01, Protocol 05, Sections 4 and 5.	Combination of nicotine replacement therapy (NRT) and medication; withdrawal management; trigger mapping; behavioural disruption.	The first 72 hours are the most acute. Week 1 is the most difficult. After Week 2, the physical withdrawal is largely resolved. The remaining work is behavioral and psychological. MAT should run for 12 weeks minimum — varenicline and bupropion are both 12-week courses.
Biological Foundation (Week 1 onwards)	Protocol 07, Protocol 09, Protocol 17	Methylation stack; choline support; NAC; pulmonary antioxidants; sleep protocol.	NAC on Day 1 — mucolytic AND craving reduction AND glutathione restoration for the oxidative debt from smoking. Alpha-GPC and choline support the cognitive function that nicotine's nAChR activation was maintaining.
Psychiatric and Underlying Condition Track (Week 1 onwards)	Psychiatry; ADHD assessment; depression treatment.	Comorbid condition management; bupropion dual use; 5-MTHF for MTHFR-depression overlap.	For 70–90% of people with psychiatric comorbidities: cessation without psychiatric management of the comorbidity almost invariably fails. The cigarettes were doing something real. The psychiatric condition needs a non-tobacco management strategy established BEFORE the tobacco is removed.
Natural Architecture Building (Months 1–3)	Protocol 03 (exercise); Protocol 04; Section 6	Exercise daily; breathwork practice; cognitive supplement stack; social replacement; mood management.	The exercise program is the most impactful single behavioural intervention for nicotine cessation, apart from Medication-Assisted Treatment (MAT). Daily aerobic exercise during cessation has been

			shown to double cessation success rates in some studies. Start with Week 1. Gradually increase intensity.
Long Game (Months 3–12)	Continuare la terapia sostitutiva con oppioidi (MAT) secondo le indicazioni del medico; rinforzo comportamentale.	Consolidation of abstinence; management of withdrawal symptoms similar to post-acute withdrawal syndrome (PAWS) including anxiety and mood fluctuations; addressing any remaining triggers.	Il rischio di ricaduta rimane elevato per 12 mesi dopo l'interruzione, in particolare durante i momenti di stress emotivo. Il protocollo di interruzione delle voglie deve rimanere accessibile e praticato. La stragrande maggioranza delle persone che mantengono l'interruzione per 12 mesi la mantiene permanentemente.

Section 8 — The Medical Context

8.1 — Medical Monitoring in Nicotine Recovery

- Función pulmonar (espirometría): si hay EPOC o síntomas respiratorios importantes. El valor inicial establece la trayectoria de mejora, lo cual es motivador para continuar con el abandono del hábito.
- Cardiovascular: blood pressure, lipid panel, echocardiogram if indicated. Cardiovascular improvement from cessation is rapid and measurable.
- Oral health: dental evaluation. Tobacco causes significant gum disease, tooth staining, and oral tissue changes.
- Screening para el cáncer: se recomienda la tomografía computarizada (TC) de detección del cáncer de pulmón para fumadores empedernidos (20 o más paquetes-año) de entre 50 y 80 años. Consulte con su médico de atención primaria.
- Thyroid: Exposure to cyanide from tobacco can affect thyroid function. A thyroid assessment is recommended for anyone experiencing related symptoms.

8.2 — Resources

- Smokefree.gov: provides free smoking cessation support, including text message coaching (send "QUIT" to 47848), live chat, and a mobile app.
- 1-800-QUIT-NOW: gratuita línea de ayuda para dejar de fumar disponible en los 50 estados; se puede ofrecer asesoramiento y terapia de reemplazo nicotínico gratuitos.
- Prescriber: Any primary care provider can prescribe varenicline and bupropion. Ask specifically.
- Nicotine Anonymous: nicotineanonymous.org — a peer support community for quitting nicotine.
- Allen Carr's Easyway: the most evidence-supported bibliotherapy approach for nicotine cessation.

A Final Word

THE CENTURION — La dipendenza più comune tra gli esseri umani ha il trattamento migliore disponibile. Usalo.

In 2022, 8 million people died from tobacco use. Currently, 1.3 billion people are dependent on nicotine. Medicines that help people stop smoking exist, are available, are covered by most insurance plans, and are more effective than almost any treatment for any other chronic condition in medicine. And the majority of people who want to quit attempt it without medication—without varenicline, without NRT, without bupropion—and fail, and conclude that they cannot quit.

They cannot quit using willpower alone. The neurobiology of nicotine dependence is real and significant. It is not a character failing that the dopamine system adapted to 20 cigarettes a day. It is not a character failing that the nAChR population upregulated around chronic nicotine exposure. These are predictable biological adaptations to a powerfully addictive molecule delivered by the fastest available CNS delivery mechanism.

The recovery framework is clear. The medicine is available. The genomics are understood — CHRNA5 A allele carriers respond best to varenicline; CYP2A6 slow metabolizers get more nicotine per cigarette and somewhat easier cessation; MTHFR variant carriers need 5-MTHF and targeted mood support during cessation; SLC6A4 short/short carriers need anxiety management built into the cessation plan. The precision is available.

And the natural recovery architecture — the exercise that replaces nicotine's dopamine, the breathwork that replaces the anxiety-reducing effect of cigarettes, the choline support that replaces the cognitive enhancement provided by nAChRs — is also readily accessible.

8 million people per year. The medicine exists. The biology is understood. This guide was built so that number can be smaller.

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Smokefree.gov | 1-800-QUIT-NOW | Nicotine Anonymous: nicotineanonymous.org

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.